COPYRIGHT (C) 1999 AMERICAN CHEMICAL SOCIETY (ACS) SINCE FILE TOTAL ******************************* FILE 'CAPLUS' ENTERED AT 15:59:45 ON 27 DEC 1999 USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER COPYRIGHT (C) 1999 Elsevier Science B.V. All rights reserved. 0.15 FILE 'INPADOC' ENTERED AT 15:59:45 ON 27 DEC 1999 COPYRIGHT (C) 1999 European Patent Office, Vienna (EPO) FILE MEDLINE' ENTERED AT 15:59:45 ON 27 DEC 1999 FILE EMBASE' ENTERED AT 15:59:45 ON 27 DEC 1999 158 RECEPTOR FOR ADVANCED GLYCATION FILE BIOSIS' ENTERED AT 15:59:45 ON 27 DEC 1999 COPYRIGHT (C) 1999 BIOSIS(R) FILE 'HOME' ENTERED AT 15:59:32 ON 27 DEC 1999 PLEASE SEE "HELP USAGETERMS" FOR DETAILS. 0.15 SESSION => s receptor for advanced glycation end?/ab,bi >> file medline embase biosis inpadoc caplus ENTRY 'AB' IS NOT A VALID FIELD CODE
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an isolated peptide having an amino acid sequence corresponding to
                                                                                                                                                                                                                                                                                                                                                                                                                                                       product, and the peptide or agent is present in an amt. effective to inhibit interaction of the ***amyloid*** -beta peptide with the ***for ***advanced*** ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       DUPLICATE 1
                                                                                                                                                                                  -.beta peptide with a ***receptor*** for ***advanced***
***glycation*** ***end*** product which is on the surface
                                                                                                                                                                                                                                                                                                                               equiv. agent, wherein the peptide or agent is capable of inhibiting
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 Helsinki, Finland.. Henri.Hutunen@helsinki.fi
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Jul 9) 274
(28) 19919-24.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        the transcription factor NF-kappaB. However, the RAGE signaling
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              Department of Biosciences, Division of Biochemistry, University
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                products or ***amyloid*** beta-peptide, is suggested to play a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       products (RAGE)-mediated neurite outgrowth and activation of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         for ***advanced*** ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               ***Receptor*** for ***advanced*** ***glycation***
                                                                                                                                                                                                                                                                                                                                                               interaction of the ***amyloid*** - beta peptide with the ***receptor*** for ***advanced*** ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        substrates. Ligation of RAGE by two other ligands, advanced
                                                               amino acid sequence of a V-domain of RAGE. The present
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 require the cytoplasmic domain of the receptor but different
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                                                                                                                                                                                                                                                                            which comprises contacting the cell with the peptide or a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              products (RAGE) mediates neurite outgrowth in vitro on
                                                                                                                       provides for a method for inhibiting interaction of an
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      AU Huttunen H J; Fages C; Rauvala H
CS Laboratory of Molecular Neurobiology, Institute of
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DT Journal; Article; (JOURNAL ARTICLE)
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FS Priority Journals; Cancer Journals
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                                                                                                                                                                                                               ***glycation***
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                                                                                                                                                       ***amyloid***
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PA The Trustees of Columbia University In the City of New York,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      AB The present method provides for an isolated peptide having an
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         RW. AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    ***glycation*** ***endproduct*** ) for therapeutic use
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        ***receptor*** for ***advanced*** ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   product (RAGE). The present invention also provides for an
                                                                                                                                                                                                                                                               21 DUP REM L5 (23 DUPLICATES REMOVED)
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DN 130:301683
TI Ligand-binding site of RAGE ( ***receptor***
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pathways	***amvloidoses*** . RAGE also functions as a signal	CNS Research
in neurite outgrowth and cell injury are largely unknown. Here we	transduction receptor	Basel CH-4070, Switzerland. parichehr.malherbe@roche.com
wows	for ****amyloid*** beta peptide, known to accumulate in	SO Molecular Brain Research, (1999) 71/2 (159-170).
that transfection of RAGE to neuroblastoma cells induces extension	Alzheimer	Refs: 26
100 Inc. Inc.	disease in both affected brain parenchyma and cerebral vasculature.	ISSN: 0169-328X CODEN: MBREE4
inopodia and neurites on amphoterin-coated substrates.	Interaction of KAGE with these ligands enhances receptor	
Fullication of BACE in transferred rells enhances	expression and initiates a manifixe feedback loop inhereby secession secures.	CY Netherlands
NF-kappaB-dependent	minates a positive recurata tody witeredy receptor occupantly	FS (M) Memplow and Memoriment
transcription. Both the RAGE-mediated neurite outgrowth and	increased RAGE expression, thereby perpetuating another wave of	0.29 Clinical Biochemistry
activation of	cellular	
NF-kappaB are blocked by deletion of the cytoplasmic domain of	activation. Sustained expression of RAGE by critical target cells,	SL English
RAGE.	including endothelium, smooth muscle cells, mononuclear	
Moreover, dominant negative Rac and Cdc42 but not dominant	phagocytes, and	***glycation***
negative Ras	neurons, in proximity to these ligands, sets the stage for chronic	***end*** products (RAGE) has been proposed as a cell surface
inhibit the extension of neurites induced by RAGE-amphoterin	cellular activation and tissue damage. In a model of accelerated	receptor
interaction.	atherosclerosis associated with diabetes in genetically manipulated	that binds *** amyloid*** - beta. protein (A. beta.), thereby
In contrast, the activation of NF-kappaB is inhibited by dominant	mice,	triggering
negative	blockade of cell surface RAGE by infusion of a soluble, truncated	its cytotoxic effects [S.D. Yan, X. Chen, J. Fu, M. Chen, H. Zhu,
Ras but not Rac or Cdc42. These data suggest that distinct signaling	form of	A.
pathways are used by RAGE to induce neurite outgrowth and	the receptor completely suppressed enhanced formation of vascular	Roher, T. Slattery, L. Zhao, M. Nagashima, J. Morser, A. Migheli,
regulate gene	lesions.	Δ.
expression through NF-kappaB.	Amelioration of atherosclerosis in these diabetic/atherosclerotic	Nawroth, D. Stern, A.M. Schmidt, RAGE and ***amyloid***
	animals	beta. peptide
L6 ANSWER 3 OF 21 MEDLINE DUPLICATE 2	by soluble RAGE occurred in the absence of changes in plasma	neurotoxicity in Alzheimer's disease, Nature 382 (1996) 685-691.].
AN 1999182371 MEDLINE	lipids or	A cDNA
	glycemia, emphasizing the contribution of a lipid- and	library of human lung was screened for RAGE with an appropriate
11 Activations of the coptoff of the advanced for the contract of the contract	gycemia-independent mechanism(s) to amerogenesis, which we	hybridization probe. In addition to cell surface KAGE, one clone
#### price of respectations of the price of	postulate to be interesting of DACE with its liceards. Disting studies miss.	was jound
	of interaction of reserve with its rigation, future statutes using mice	which encodes a new version of KAGE, termed hkaGEsec, which
diabetic vascuilonathy and atheroselemeis	III urkich DACE everection has been censtically manipulated and with	index sure 19
AU Schmidt AM: Yan SD: Wautier J1:: Stem D	selective	annio avias of alc memorapannig region and is arrenote
CS Division of Surgical Science, Department of Surgery, College of	low molecular weight RAGE inhibitors will be required to	Comparison with the penomic sequence revealed that the synthesis
Physicians	definitively	of the
& Surgeons of Columbia University, New York, NY 10032, USA.	assign a critical role for RAGE activation in diabetic vasculonathy.	secreted isoform remires alternative splicing. The deduced protein
SO CIRCULATION RESEARCH, (1999 Mar 19) 84 (5) 489-97.	However, sustained receptor expression in a microenvironment	sequence of the mature hRAGEsec consists of 321 amino acids
Ref. 89	with a	with a
Journal code: DAJ. ISSN: 0009-7330.	plethora of ligand makes possible prolonged receptor stimulation.	predicted molecular mass of 35.66 kDa. The pattern of expression
CY United States	suggesting that interaction of cellular RAGE with its ligands could	Jo
DT Journal; Article; (JOURNAL ARTICLE)	De a	hRAGEsec in human brain was analyzed by in situ hybridization
General Review, (REVIEW)	factor contributing to a range of important chronic disorders.	histochemistry. The most intense expression of the gene in contrast
(REVIEW, TUTORIAL)		to
LA English	L6 ANSWER 4 OF 21 EMBASE COPYRIGHT 1999 ELSEVIER	cell surface RAGE was detected in hippocampal CA3 pyramidal
FS Priority Journals	SCI. B.V.DUPLICATE 3	cells, dentate
EIVI 199903	AN 19993003/3 EMBASE	gyrus granule cells, cortical neurons as well as glial cells in white
一年 日本の日本の日本の日本の日本の日本の日本の日本の日本の日本の日本の日本の日本の日	11 cUNA cioning of a novel secreted isoform of the human	matter. To investigate the interaction between A.beta. and RAGE
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wind the comment of the comment of the immunity of the immunit	Bridgerion	anounci scavenger receptor, SRA, under physiological conditions,
produces (1990) is a memora of the minimal growning superioring)	characterization of cells co-expressing cell-surface scavenoer	LICY WELE COLEVERSESED With himon beta ADD605.SEAD in a himon cell and
surface molecules and engages diverse ligands relevant to distinct	receptors	the level of
pathological processes. One class of RAGE ligands includes	and Swedish mutant ***amyloid*** precursor protein.	A.beta. in the condition medium was assessed by
glycoxidation	AU Malherbe P.; Richards J.G.; Gaillard H.; Thompson A.; Diener	immunoprecipitation and
products, termed advanced glycation end products, which occur in	C.; Schuler	enzyme-linked immunosorbent assay (ELISA) analysis. A nearly
diabetes,	A; Huber G.	%001
at sites of oxidant stress in tissues, and in renal failure and	CS P. Malherbe, Pharma Division PRPN, Bldg, 69/333, Preclinical	reduction of A.beta. from the conditioned medium of hRAGE cells

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The ***receptor*** for ***advanced***
glycation***
***end*** products (RAGE) has been proposed as a cell surface
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    s cytotoxic effects [S.D. Yan, X. Chen, J. Fu, M. Chen, H. Zhu,
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CNS Research,
Basel CH-4070, Switzerland, pariothehr malherbe@roche.com
SO Molecular Brain Research, (1999) 71/2 (159-170).
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ISSN: 0169-328X CODEN: MBREE4
PUI S 0169-328X(99)00174-6
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  nat binds ***amyloid*** ..beta. protein (A.beta.), thereby
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I Journal; Article
008 Neurology and Neurosurgery
029 Clinical Biochemistry
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dependent, polarized to the apical side, and saturable with high- and low-affinity dissociation constants of 7.8+/-1.2 and 52.8+/-6.2 nM, or macrophage scavenger receptor (SR), type A, displayed binding SO JOURNAL OF CLINICAL INVESTIGATION, (1998 Aug 15) CS Department of Neurological Surgery, USC School of Medicine, binding to the brain microvascular endothelial cell monolayer was blockade (36%) but not to SR blockade. We conclude that RAGE with a Michaelis constant of 45+/-9 nM, and partially sensitive to neurotoxicity if it crosses the brain capillary endothelium, which mediate binding of sAbetal 40 at the apical side of human BBB, AB A soluble monomeric form of Alzheimer's ***amyloid*** Abridged Index Medicus Journals; Priority Journals; Cancer ***receptor*** for ***advanced*** ***glycation*** intact > 94%. Transcytosis of 1251-sAbetal 40 was time and comprises the blood-brain barrier (BBB) in vivo. This study internalization of 1251-sAbetal 40. The internalized peptide peptide (sAbetal 40) is present in the circulation and could dependent, asymmetrical from the apical to basolateral side, respectively. Binding of 1251-sAbetal-40 was inhibited by human sAbetal 40 using an in vitro model of human BBB. endothelial binding and transcytosis of a synthetic peptide products) antibody (63%) and by acetylated low density Consistent with these data, transfected cultured cells Stern D; Schmidt A M; Frangione B; Zlokovic B V RAGE is also involved in sAbetal 40 transcytosis Journal; Article; (JOURNAL ARTICLE) Journal code: HS7, ISSN: 0021-9738. California 90033, USA NC NS-34467 (NINDS) overexpressing RAGE AG-14526 (NIA) AG-05891 (NIA) CY United States lipoproteins (33%) 1251-sAbeta1 40 19981103 102 (4) 734-43. nomologous to EM 199811 contribute to characterizes anti-RAGE (English -beta (1-40) temperature ...buo Journals saturable and SR RAGE F Y for ***advanced*** ***glycation*** ***end*** products **DUPLICATE 4** receptors and Swedish mutant ***amyloid*** precursor protein. SO Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp. SO Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp. CS Pharma Div., Preclinical CNS Res., F. Hoffmann-La Roche Ltd., AU Mackic J B; Stins M; McComb J G; Calero M; Ghiso J; Kim K and characterization of cells co-expressing cell-surface scavenger 1-40. Asymmetrical binding, endocytosis, and transcytosis at the ***Amyloid*** -beta peptide: Structure and neuro-toxicity. .apprx.40% reduction from the SRA-cells implied that hRAGE AU Malherbe, P.; Richards, J. G.; Gaillard, H.; Thompson, A.; L6 ANSWER 5 OF 21 BIOSIS COPYRIGHT 1999 BIOSIS L6 ANSWER 6 OF 21 BIOSIS COPYRIGHT 1999 BIOSIS Eisai Tsukuba Res. Lab., Eisai Co. Ltd., Tsukuba, Ibaraki AU Hashimoto, T.; Omac, H.; Kobayashi, K.; Miyagawa, T.; cDNA Cloning of a novel secreted isoform of the human Nakagawa, M.; Kuwada, M.; Ogura, H.; Nishizawa, Y. prominent cell surface receptor interacting with A.beta. Meeting Info.: 28th Annual Meeting of the Society for Meeting Info.: 28th Annual Meeting of the Society for side of brain microvascular endothelial cell monolayer. TI Human blood-brain barrier receptors for Alzheimer's Los Angeles, California, USA November 7-12, 1998 Los Angeles, California, USA November 7-12, 1998 L6 ANSWER 7 OF 21 MEDLINE AN 1998376482 MEDLINE DN 98376482 AN 1999:80034 BIOSIS DN PREV199900080034 DN PREV199900081096 1999:81096 BIOSIS Schuler, A.; Huber, G. ***amyloid*** -beta Basel Switzerland ISSN: 0190-5295. Neuroscience, Part 2 ISSN: 0190-5295. Neuroscience, Part 2 Conference Conference 300-2635 Japan Watanabe, T.: LA English LA English S; Yan S D; Diener, C.; CH 4575 <u>4</u>6 <u>8</u> S 占 Д

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SO Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp.
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             Characterization of the ***receptor*** for ***advanced***
***glycation*** ***endproducts*** (RAGE) in brain
                                                                                                                                                                       AU Miao, W. (1); Mackic, J. B.; Ghiso, J.; Yamada, S.; Jovanovic,
                                                                                                                                                                                                                                                                                                                                                     CS (1) Dep. Neurosurgery, USC Sch. Med., Children's Hosp. L.A.,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     AU Lue, L.-F. (1); Shen, Y. (1); Yan, S.; Stem, D.; Rogers, J. (1) CS (1) Roberts Alzheimer's Res. Cent., Sun Health Res. Inst., Sun
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 Los Angeles, California, USA November 7-12, 1998 Society for
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                                                                           TI Rage mediates in vivo transport of Alzheimer's Abetal 40 and
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AN 1999:52634 BIOSIS
DN PREV199900052634
                                                                                                                                                                                                                                            J. G., Van Nostrand, W., Yan, S. D., Fragione, B., Stem, D.,
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PA Trustees of Columbia University, USA
                                                                                                                                       peptides at the blood-brain barrier in rodents.
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cell receptor for ***amyloid*** beta protein (A beta). To determine if this is indeed the case, two neural cell lines as well as rat cortical neurons E4. A beta. induces expression of tau protein kinase I (TPK-I) in cultured nerve cells, and the antisense oligonucleotide of TPK-I suppresses neurotoxicity of A beta. Protein kinase C is induced or mRNA detected in the cultured neural cells. Glycated-albumin is a neurotoxicity of A.beta. by apolipoprotein E are by the order of E2 were examined for the presence of the mRNA for RAGE by PCR blot analysis. Although lung was strongly positive, in no case was ***glycation*** ***end*** products (RAGE) is the nerve agreement with the mRNA data, trypsin treatment did not alter A toxicity, nor did glycated albumin modify the A beta response. It AU Yanagisawa, Katsuhiko CS Natl. Inst. Longevity Sci., Natl. Chubu Hosp., Obu, 474, Japan SO Dementia Jpn. (1997), 11(1), 34-42 free radical generation. The suppressing activity of oxygen stress beta -protein (A beta.) discussed; the toxicity closely correlates CS The Salk Institute for Biological Studies, La Jolla California ligand for RAGE and the cell surface RAGE protein is trypsin ANSWER 14 OF 21 CAPLUS COPYRIGHT 1999 ACS BIOCHEMICAL AND BIOPHYSICAL RESEARCH AB A review with 33 refs. Neurotoxic mechanisms of AB It has been suggested that a ***receptor*** that RAGE is not the neural receptor for A beta COMMUNICATIONS, (1997 Aug 8) 237 (1) Journal; Article; (JOURNAL ARTICLE) TI Neurotoxicity of A.beta,- ***amyloid*** CODEN: DEJAFB; ISSN: 1342-646X Journal code: 9Y8. ISSN: 0006-291X. FS Priority Journals; Cancer Journals EM 199711 EW 19971102 L6 ANSWER 14 OF 21 CAI AN 1997:264711 CAPLUS DN 126:315587 DT Journal; General Review NC NS09658 (NINDS) NS28121 (NINDS) NS10279 (NINDS) CY United States PB Esu Ato K.K. ***advanced*** ***amyloid*** LA Japanese English and northern sensitive. In 3740 ΓĄ CS (1) Dep. Pathology, Columbia Univ., New York, NY 10032 USA Abeta-stimulated neurons interacts with its cognate receptor, c-fms, TI Rage and A-beta in Alzheimer disease (AD): Cell surface receptor AU Yan, S. D.; Levine, H. (1); Soto, C.; Zhu, A.; Zhu, H.; Chen, X.; cerebrospinal fluid might provide a means for monitoring neuronal SO Society for Neuroscience Abstracts, (1997) Vol. 23, No. 1-2, pp. triggered by engagement of Abeta on neuronal RAGE. We suggest DUPLICATE AN 97410110 MEDLINE DN 97410110 TI Beta ***amyloid*** toxicity does not require RAGE protein. antigen (P < 0.01), compared with age-matched controls. M-CSF enhanced survival of microglia exposed to Abeta, consistent with pathologic findings in AD. These data delineate an inflammatory ***Endproduct*** (RAGE), a cell surface receptor for Abeta fluid from AD patients there was approximately 5-fold increased binding of ***amyloid*** -beta peptide (Abeta) to neuronal ***Receptor*** for ***Advanced*** ***Glycation*** thus generated, contributes to the pathogenesis of AD, and that macrophage-colony stimulating factor (M-CSF) by an oxidant ANSWER 12 OF 21 BIOSIS COPYRIGHT 1999 BIOSIS expression of M-CSF in proximity to Abeta deposits, and in nuclear factor kappaB-dependent pathway. AD brain shows microglia, thereby triggering chemotaxis, cell proliferation, deleterious cytotoxic effects of activated microglia. We Meeting Info.: 27th Annual Meeting of the Society for expression of the macrophage scavenger receptor and fibrils and soluble receptor prevents fibrillogenesis. Orleans, Louisiana, USA October 25-30, 1997 Conference; Abstract; Conference perturbation at an early stage in AD. ANSWER 13 OF 21 MEDLINE AU Liu Y; Dargusch R; Schubert D A.; Stem, D.; Schmidt, A. M. AN 1997:531767 BIOSIS DN PREV199799830970 apolipoprotein E, and ISSN: 0190-5295 Neuroscience New increased neuronal that M-CSF. released by M-CSF in M-CSF 2 2 5 identify the interaction of .beta.- ***amyloid*** with RAGE are EM 199708 AB In Alzheimer disease (AD), neurons are thought to be subjected products) in neural cells and induces neurotoxic damage typical of DUPLICATE AU 1997-18327 19970121 and characterization of .beta. - ***amyloid*** -binding proteins AU Du Yan S; Zhu H; Fu J; Yan S F; Roher A; Tourtellotte W W; RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, Alzheimer's disease. This interaction may be a useful target for AB The beta. ***amyloid*** protein binds to a cell-surface ***receptor*** for ***advanced*** ***glycation*** APPLICATION NO CS Department of Pathology, Columbia University, College of described. Peptides capable of inhibiting the interaction are ***glycation*** ***endproduct*** interaction elicits SO PROCEEDINGS OF THE NATIONAL ACADEMY OF treatment of Alzheimer's disease. Binding assays for the ***Amyloid*** -beta peptide- ***receptor*** for WO 1997-US857 expression of macrophage-colony stimulating factor: a Chen X; Godman G C; Stem D; Schmidt A M AMERICA, (1997 May 13) 94 (10) 5296-301 Journal; Article; (JOURNAL ARTICLE) SCIENCES OF THE UNITED STATES OF Surgeons, New York, NY 10032, USA Journal code: PV3. ISSN: 0027-8424. LA English FS Priority Journals; Cancer Journals Al 19970731 L6 ANSWER 11 OF 21 MEDLINE Al 19970820 KIND DATE PRAI US 1996-592070 19960126 pathway in Alzheimer disease. WO 1997-US857 19970121 AN 97289760 MEDLINE W: AU, CA, JP, MX NC AG00690 (NIA) PI WO 9726913 AG00602 (NIA) AG11925 (NIA) CY United States PATENT NO. ***advanced*** MC, NL, PT, SE proinflammatory AU 9718327 DN 97289760 Rajavashisth T. Physicians and identification ***end***

reported.

RAGE (

neuronal

tumor necrosis factor-alpha (TNF) expression by MPs exposed to 96345664 Physicians and AGE-beta2M deposits of a Alzheimer's Z mononuclear phagocytes (MPs), cells important in the pathogenesis The ***receptor*** for ***advanced*** ***glycation***
end products (RAGE) is a central mediator of the SO JOURNAL OF CLINICAL INVESTIGATION, (1996 Sep 1) 98 AGE-beta2microglobulin with human mononuclear phagocytes via association with the ***receptor*** for ****advanced***
glycation ***end*** products (RAGE) and the class versus controls. These data, coupled with recent discoveries of the DUPLICATE AU Miyata T; Hori O; Zhang J; Yan S D; Ferran L; Iida Y; Schmidt glycation end products (AGEs) of the Maillard reaction, known as inflammatory arthropathy of dialysis-related ***amyloidosis*** mediated by the receptor for AGEs, or RAGE. 1251-AGE-beta2M ***amyloidosis*** is a form of beta2microglobulin modified approximately 53.5 and approximately 81.6 nM, respectively), a chemotaxis was prevented by excess sRAGE or anti-RAGE IgG. oxidant-sensitive pathway. Implications for the pathogenesis of receptors (SR), support the hypothesized role of inflammatory CS Department of Internal Medicine, Branch Hospital, Nagoya Abridged Index Medicus Journals; Priority Journals; Cancer immobilized RAGE or to MPs in a specific, dose-dependent AGE-beta2M. We demonstrate here that the interaction of An important component of ***amyloid*** fibrils in inhibited in the presence of RAGE blockade. Journal; Article; (JOURNAL ARTICLE) Journal code: HS7, ISSN: 0021-9738 dialysis-related ***amyloidosis*** L6 ANSWER 16 OF 21 MEDLINE AGE-beta2M-mediated monocyte AN 96379656 MEDLINE in AD neurotoxicity. of Medicine, Japan. HL21006 (NHLBI) NC AG00602 (NIA) United States AGE-beta2M with University School 19970104 96379656 dialysis-related with advanced interaction of EM 199701 (5) 1088-94. Journals of the CY П FS and astrocytes, in turn, form a functional barrier between A beta and A.beta. generates oxygen stress and secretion of cytotoxic cytokines disease (AD). Rather than exerting its neurotoxicity directly, results Department of Psychiatry, University of Minnesota, Minneapolis, suggest that beta- ***amyloid*** (A beta) is directly involved in surrounding neurons. An increase in inducible nitric oxide synthase immunoreactivity is observed in activated microglia and astrocytes, from our laboratory suggest that fibrillar A beta (fA beta) activates suppressed by A.beta. depending on the concn. A.beta. suppresses activity of phosphatidylinositol 4-kinase and binding of fibronectin A.beta. forms a pore in membrane by hexamer formation with hair of intracellular Ca conen. by glutamic acid or calcium ionophore, AB Several lines of evidence, including newly discovered genetic structure. A.beta. induces functional anomaly in K channel, and specific subpopulations of neurons are lost in fA beta injection microglia and astrocytes upon injection into the rat brain. The tumor necrosis factor .alpha. (TNF alpha.) through binding to cells. A beta elevates intracellular Ca conen., and enhances secretion of cytokines in some cell lines. A.beta. suppresses neuropathology observed in familial and sporadic forms of DN 97452789 TI New insights into the neuropathology and cell biology of SO GERIATRICS, (1997 Sep) 52 Suppl 2 S13-6. Ref: 12 Journal code: FOI. ISSN: 0016-867X. ubiquitin-dependent protein degran. by inhibition of 26S Abridged Index Medicus Journals; Priority Journals product (RAGE) and scavenger receptor (SR) Journal; Article; (JOURNAL ARTICLE) AU Weldon DT; Maggio JE; Mantyh P W L6 ANSWER 15 OF 21 MEDLINE General Review; (REVIEW) AN 97452789 MEDLINE REVIEW, ACADEMIC) United States LA English FS Abridged EM 199712 ***endo*** proteasome. mutations

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AGE-beta2M reduced cytochrome c and the elaboration of TNF by
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 MPs infiltrating these lesions. These data indicate that RAGE is a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    DUPLICATE
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                a process which may ultimately lead to bone and joint destruction.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     AU Yan S D; Chen X; Fu J; Chen M; Zhu H; Roher A; Slattery T;
                                                                                            TNF antigen release into culture supernatants were prevented by
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               site that could focus its effects on target cells has been postulated
                                                                                                                                                                                of sRAGE, a process mediated, at least in part, by oxidant stress.
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                                                                                                                                                                                                                                                                                                                          inhibited by N-acetylcysteine. Consistent with these data, immunohistochemical studies of AGE-laden ***amyloid****
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                TI RAGE and ***amyloid*** -beta peptide neurotoxicity in
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       disease, because it is neurotoxic-directly by inducing oxidant
resulted from engagement of RAGE, as appearances of TNF
                                                                                                                                                                                                                                                                                                                                                                                                                                                                         long-term hemodialysis patient revealed positive staining for
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               and indirectly by activating microglia. A specific cell-surface
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         binding site for AGEs formed in vivo and suggest that
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 disease [see comments].

CM Comment in: Nature 1996 Aug 22;382(6593):674
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SO NATURE, (1996 Aug 22) 382 (6593) 685-91.
Journal code: NSC. ISSN: 0028-0836.
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Journal; Article; (JOURNAL ARTICLE)
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FS Priority Journals; Cancer Journals
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nude on (DR) heimer's Stem ronal CS ronal CS Nage Nage No SO	(AGEs): Role in the pathogenesis of dialysis-related ***amyloidosis*** (DRAU lida, Yoshiyasu; Miyata, Toshio; Maeda, Kenji; Hori, Osamu; Stem, David; Schmidt, Ann M. CS Dep. Inter. Med., Branch Hosp., Nagoya Univ. Sch. Med., Nagoya Japan CS Dep. Inter. Med., Branch Hosp., Nagoya Univ. Sch. Med., No. 3, pp. 536. Meeting Info:: Annual Meeting of the American Society of Nephrology San Diego, California, USA November 5-8, 1995 ISSN: 1046-6673. DT Conference LA English L6 ANSWER 21 OF 21 BIOSIS COPYRIGHT 1999 BIOSIS AN 1995:8108 BIOSIS DN PREV199598022408 TI The mononuclear phagocyte interaction site of beta-2-microgolobulin modified by glycation is the ***receptor*** for ***earlycation*** ***endproducts*** ***elycation*** ***endproducts*** ***abavanced**** ***Bavohi; Stem. David; Miyata, Toshio CS (1) Columbia Univ., New York, NY USA SO Circulation, (1994) Vol. 90, No. 4 PART 2, pp. 1233. Meeting Info:: 67th Scientific Sessions of the American Heart	=> s 7 and 1 L8 31 L7 AND L1 => dup rem 8 PROCESSING COMPLETED FOR L8 L9 24 DUP REM L8 (7 DUPLICATES REMOVED) => s 8 and presen?/dab, bi 'AB' IS NOT A VALID FIELD CODE 'AB' IS NOT A VALID FIELD CODE 'AB' IS NOT A VALID FIELD CODE 'AB' IS NOT A VALID FIELD CODE 'AB' IS NOT A VALID FIELD CODE 'AB' IS NOT A VALID FIELD CODE 'AB' IS NOT A VALID FIELD CODE 'AB' IS NOT A VALID FIELD CODE 'AB' IS NOT A VALID FIELD CODE L10 9 L8 AND PRESEN?/AB,BI => dup rem 10 PROCESSING COMPLETED FOR L10 L11 7 DUP REM L10 (2 DUPLICATES REMOVED) => d 1- bib ab YOU HAVE REQUESTED DATA FROM 7 ANSWERS - CONTINUE? Y(N):y L11 ANSWER I OF 7 CAPLUS COPYRIGHT 1999 ACS AN 1999:691229 CAPLUS DN 131:317761 TI Inhibition of fumor invasion or spreading based on a soluble ***********************************
Associal ***Auryloid**** - beta peptide-induced activation of microglia. AU Yan, Shi-Du (1); Chen, Xi; Fu, Jin; Chen, Ming, Zhu, Huaijie; AU Yan, Shi-Du (1); Chen, Xi; Fu, Jin; Chen, Ming, Zhu, Huaijie; Au Yan, Shi-Du (1); Chen, Xi; Fu, Jin; Chen, Ming, Zhu, Huaijie; Au Yan, Shi-Du (1); Chen, Xi; Fu, Jin; Chen, Ming, Zhu, Huaijie; Ngashima, Mariko; Morser, John; Roher, Alex; Stem, David; Schmidt, Ann Marie CS (1) Columbia Univ., New York, NY 10032 USA Schoiety for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. E1 194. Meeting Info:. 26th Amual Meeting of the Society for E3 Neuroscience Washington, D.C., USA November 16-21, 1996 ISSN: 0190-5295. LA Finglish LA AN 1996-5210 BIOSIS TI Monocyte/macrophage interaction of nonenzymatically glycated beta-2-microglobulin (beta-2M) is mediated by the beta-2-microglobulin (beta-2M) is mediated by the **endvanced*** **evendvanced*** **evendvanced***evendvanced*** **evendvanced*** **evendvanced*** **evendvanced***evendvanced*** **eve	Association Dallas, Texas, USA November 14-17, 1994 ISSN: 00099-7322 DT Conference LA English => e stem david/au E1 1 STERN DARRYL/AU E2 221> STERN DARRYL AAU E3 221> STERN DAVIDAU E4 11 STERN DAVIDAU E5 97 STERN DAVIDAU E6 97 STERN DAVIDAU E7 2 STERN DAVID FAU E8 76 STERN DAVID FAU E8 76 STERN DAVID FAU E9 1 STERN DAVID FAU E9 1 STERN DAVID FAU E9 2 STERN DAVID FAU E10 2 STERN DAVID FAU E11 4 STERN DAVID LAU E12 78 STERN DAVID LAU E13 78 STERN DAVID LAU E>s e3	***endproducts*** IN Schmidt, Ann Marie; ***Stem, David*** PA The Trustees of Columbia University in the City of New York, USA SO PCT Int. Appl., 88 pp. CODEN: PIXXD2 DT Patent LA English FAN.CNT I PATENT NO. KIND DATE APPLICATION NO. DATE PATENT NO. ALI 19991028 WO 1999-US8427 19990416 W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CJ, CZ, CN, CJ, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TI, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TI, TM

glycation ***end*** product. receptor for \$100/calgranulin polypeptides A1 19990422 AU 9897958 A1 19990503 PRAI US 1997-948131 19971009 WO 1998-US21346 19981009 1999:434649 CAPLUS W: AU, CA, JP, MX A-O-N-1-T-A-R-1-G-E-Taguchi, Akihiko; PI WO 9918987 ***advanced*** PT. SE contacting the LU, MC, NL, also provides V-domain of method for ***end*** V-domain having an Ä environment which comprises: (a) admixing with cell culture media matrix (amphoterin and/or similar structures) interaction appears to ***present*** invention also provides a method for evaluating thus evaluating the ability of the agent to inhibit tumor invasion in with the media from step (a); (c) detg. the amt. of spreading of the invasion or metastasis in a subject which comprises administering least one mechanism by which sRAGE limits tumor growth. The cell culture, and (d) comparing the arnt. of spreading of the turnor local cellular environment. The ***present*** invention also PA The Trustees of Columbia University In the City of New York culture detd. in step (c) with the amt. detd. in the absence of the RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ ***glycation*** ***endproduct***) for therapeutic use APPLICATION NO IN ***Stem, David***; Yan, Shi Du; Schmidt, Ann Marie, subject a therapeutically effective ant. of a form of sol. ability of an agent to inhibit tumor invasion in a local cellular effective amt. of the agent; (b) contacting a tumor cell in cell PRAI US 1998-62365 19980417
AB The ***present*** invention provides for a method for a pharmaceutical compn. which comprises a therapeutically of the agent evaluated in the aforementioned method and a LII ANSWER 2 OF 7 CAPLUS COPYRIGHT 1999 ACS CI, CM, GA, GN, GW, MIL, MR, NE, SN, TD, TG ***endproducts*** (RAGE). Interruption of cellular AN 1999:265908 CAPLUS
DN 130:301683
TI Ligand-binding site of RAGE (***receptor*** for KIND DATE SO PCT Int. Appl., 101 pp. CODEN: PLXXD2 acceptable carrier. PATENT NO. RAGE-extracellular inhibiting tumor LA English FAN.CNT 1 CF, CG, 占

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TI Human blood-brain barrier receptors for Alzheimer's amyloid-beta
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        inflammation, likely released by inflammatory cells targeted to such
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             SO Journal of Clinical Investigation, (Aug. 15, 1998) Vol. 102, No.
                                                                                                                               CS College of Physicians and Surgeons, Columbia University, New
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 AB S100/calgranulin polypeptides are ***present*** at sites of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       Asymmetrical binding, endocytosis, and transcytosis at the apical
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    CS (1) USC Sch. Med., 2025 Zonal Ave., RMR 506, Los Angeles,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      for AGE (advanced glycation end products) (RAGE) is a central
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             Schmidt, Ann Marie; Fragione, Blas; Zlokovic, Berislav V. (1)
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              EN-RAGEs with cellular RAGE on endothelium, mononuclear
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        lymphocytes triggers cellular activation, with generation of key
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 by a range of environmental cues. The authors report here that
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                hypersensitivity and inflammatory colitis in murine models by
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       AU Mackic, Jasmina B.; Stins, Monique; McComb, J. Gordon;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              proinflammatory mediators. Blockade of EN-RAGE/RAGE
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                identify roles for EN-RAGEs and RAGE in chronic cellular
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       L11 ANSWER 4 OF 7 BIOSIS COPYRIGHT 1999 BIOSIS
Nagashima, Mariko; Morser, John; ***Stem, David***;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              gene mediators. These data highlight a novel paradigm in
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             activation of central signaling pathways and expression of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             and related members of the S100/calgranulin superfamily.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        receptor for EN-RAGE (extracellular newly identified
                                                                                                                                                                                                                                                            SO Cell (Cambridge, Mass.) (1999), 97(7), 889-901
CODEN: CELLB5; ISSN: 0092-8674
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                brain microvascular endothelial cell monolayer.
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  RAGE-binding protein)
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 quenches delayed-type
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             inflammation and
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                                                      Schmidt, Ann
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   amino acid sequence corresponding to the amino acid sequence of a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             of a ***receptor*** for ***advanced*** ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                ***end*** product (RAGE). The ***present*** invention
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      P-L-V-L-K-C-K-G-A-P-K-K-P-P-Q-R-L-E-W-K (Seq. ID No. 1).
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   ***present*** invention provides for a pharmaceutical compn.
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  ***present*** in an amt. effective to inhibit interaction of the
                                                                                                                                                                                                                                                                                                              AU 1998-97958 19981009
                                                                                                                                                                                                                                                                                                                                                                                                                                          AB The ***present*** method provides for an isolated peptide
                                                                                                                                                                          RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  cell with the peptide or a functionally equiv. agent, wherein the
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    or agent is capable of inhibiting interaction of the amyloid-beta.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 a therapeutically effective amt. of an isolated peptide having an
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                AU Hofmann, Marion A.; Drury, Steven; Fu, Caifeng; Qu, Wu;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                RAGE mediates a novel proinflammatory axis: a central cell
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      acid sequence corresponding to the amino acid sequence of a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        RAGE. The ***present*** invention also provides for a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          Peter, Neurath, Markus F.; Slattery, Timothy; Beach, Dale;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                L11 ANSWER 3 OF 7 CAPLUS COPYRIGHT 1999 ACS
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                                                      WO 1998-US21346
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             product which is on the surface of a cell, which comprises
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         inhibiting interaction of an amyloid..beta_peptide with a
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     for an isolated peptide having an amino acid sequence
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attraction into the vessel wall where AGEs deposit/form, suggesting CS (1) Dep. Physiology, P and S 11-518, Columbia Univ., Coll P and cells, mesangial cells, mononuclear phagocytes and certain neurons both cases, the pro-inflammatory effects of AGEs may be inhibited ***advanced*** ***glycation*** ***end*** products in Brett, Jerold; Schmidt, Ann Marie; Yan, Shi Du; Zou, Yu Shan; accumulate in the tissues during aging and at an accelerated rate in that inhibition of RAGE may interfere with monocyte chemotaxis potential of this intervention to interfere with a critical step in the which is the attraction of mononuclear phagocytes into the vessel AB Advanced glycation end products (AGEs), the final products of diabetes. A novel integral membrane protein, termed receptor for cellular oxidant stress is the enhanced expression of vascular cell ***presence*** of RAGE blockade, using either anti-RAGE soluble RAGE, the extracellular domain of the molecule. These Craig, Shaw, Alan; Migheli, Antonio; ***Stern, David (1)*** glycation and oxidation of proteins, are found in the plasma and (RAGE), forms a central part of the cell surface binding site for ***present*** on the surface of endothelial cells, smooth SO American Journal of Pathology, (1993) Vol. 143, No. 6, pp. L11 ANSWER 7 OF 7 BIOSIS COPYRIGHT 1999 BIOSIS Eliott; Pinsky, David; Nowygrod, Roman; Neeper, Michael; development of vascular disease, especially in patients with interact with RAGE, resulting in the induction of monocyte Using monospecific, polyclonal antibody raised to human adhesion molecule-1 on the endothelial surface, a critical II Survey of the distribution of a newly characterized well as oxidant stress. One of the consequences of West 168th Street, New York, NY 10032 USA AN 1994:354438 BIOSIS DN PREV199497367438 AGE-RAGE-induced ***receptor*** for ISSN: 0002-9440. recombinant and consequence of DT Article data suggest Weideman, 1699-1712. F(ab')-2 or Przysiecki diabetes. tissues. S, 630 wall Ą ***end*** -products has a central role in mediating the effects of for ***advanced*** ***glycation*** ***end*** products' The ***receptor*** for ***advanced*** ***glycation*** important in the setting of diabetes mellitus due to hyperglycaemia CS (1) Dep. Physiol., Columbia Univ. Coll. Phys. Surg., New York, characteristic of this disorder. Our work has demonstrated that one AB Amyloid-beta peptide is central to the pathology of Alzheimer's indirectly by activating microglia. A specific cell-surface acceptor principal means by which AGEs interact with the vascular wall is interaction with their cellular receptor, the ***receptor*** for ***advanced*** ***glycation*** ***end*** -products that could focus its effects on target cells has been postulated but advanced glycation end-products on the development of vascular Proteins or lipids exposed to aldose sugars undergo initial and because it is neurotoxic-directly by inducing oxidant stress, and is such a receptor, and that it mediates effects of the peptide on irreversible modification resulting in the formation of so-called Matsumoto, Masayasu; ***Stem, David***; Schmidt, Ann AU Hon, Osamu (1), Yan, Shi Du; Ogawa, Satoshi; Kuwabara, and microglia. Increased expression of RAGE in Alzheimer's L11 ANSWER 6 OF 7 BIOSIS COPYRIGHT 1999 BIOSIS SO Nephrology Dialysis Transplantation, (1996) Vol. 11, No. glycation end-products (AGEs). AGEs are postulated to be indicates that it is relevant to the pathogenesis of neuronal identified. Here we *** present *** evidence that the AN 1997:128146 BIOSIS PREV199799419959 ISSN: 0931-0509. diabetes mellitus. (RAGE), which ***receptor*** **DUPLICATE** 2 SUPPL. 5, pp. and death. English disease brain Article DT Article dysfunction NY 10032 disease in 13-16 ultimately advanced especially Keisuke; (RAGE) neurons Ñ 占 ΓĄ Ζ dependent, polarized to the apical side, and saturable with high- and CS (1) Dep. Pathol., Columbia Univ., Coll. Physicians Surgeons, 630 low-affinity dissociation constants of 7.8 +- 1.2 and 52.8 +- 6.2 nM, to neurotoxicity if it crosses the brain capillary endothelium, which or macrophage scavenger receptor (SR), type A, displayed binding with a Michaelis constant of 45 +- 9 nM, and partially sensitive to binding to the brain microvascular endothelial cell monolayer was blockade (36%) but not to SR blockade. We conclude that RAGE AU Yan, Shi Du (1); Chen, Xi; Fu, Jin; Chen, Ming; Zhu, Huaijie; Slattery, Timothy; Zhao, Lei; Nagashima, Mariko; Morser, John; A soluble monomeric form of Alzheimer's amyloid-beta (1-40) Antonio; Nawroth, Peter, ***Stern, David***; Schmidt, Ann mediate binding of sAbetal 40 at the apical side of human BBB, RAGE and amyloid-beta peptide neurotoxicity in Alzheimer's (sAbeta1-40) is ***present*** in the circulation and could ***receptor*** for ***advanced*** ***glycation*** internalization of 1251-sAbetal 40. The internalized peptide intact > 94%. Transcytosis of 1251-sAbetal 40 was time and SO Nature (London), (1996) Vol. 382, No. 6593, pp. 685-691 L11 ANSWER 5 OF 7 BIOSIS COPYRIGHT 1999 BIOSIS comprises the blood-brain barrier (BBB) in vivo. This study dependent, asymmetrical from the apical to basolateral side, respectively. Binding of 1251-sAbeta1-40 was inhibited by human sAbetal 40 using an in vitro model of human BBB. endothelial binding and transcytosis of a synthetic peptide products) antibody (63%) and by acetylated low density Consistent with these data, transfected cultured cells RAGE is also involved in sAbetal 40 transcytosis 168th St., New York, NY 10032 USA 1996:425030 BIOSIS DN PREV199699156086

overexpressing RAGE

and SR

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ipoproteins (33%)

anti-RAGE (***bua***

1251-sAbetal 40 homologous to characterizes

ISSN: 0028-0836

an isolated peptide having an amino acid sequence corresponding to ***glycation*** ***end*** product, and the peptide or agent Kislinger, Thomas, Fu, Caifeng, Huber, Birgit, Qu, Wu; Taguchi, sequence corresponding to the amino acid sequence of a V-domain provides for a method for inhibiting interaction of an amyloid-beta for a pharmaceutical compn. comprising a therapeutically effective wherein the peptide or agent is capable of inhibiting interaction of II N.epsilon.-(carboxymethyl)lysine adducts of proteins are ligands AB The present method provides for an isolated peptide having an contacting the cell with the peptide or a functionally equiv. agent, RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, products that activate cell signaling pathways and modulate gene CS College of Physicians & Surgeons, Columbia University, New ***end*** product which is on the surface of a cell, which ***receptor*** for ***advanced*** ***glycation*** ***receptor*** for ***advanced*** ***glycation*** product (RAGE). The present invention also provides for an amino acid sequence of a V-domain of RAGE. The present A-P-K-K-P-P-Q-R-L-E-W-K (Seq. ID No. 1). The present L14 ANSWER 2 OF 20 CAPLUS COPYRIGHT 1999 ACS peptide with the ***receptor*** for ***advanced*** ***Yan, Shi Du***; Hofmann, Marion; Yan, Shi Fang, peptide with a ***receptor*** for ***advanced*** amyloid-beta peptide with the ***receptor*** for present in an amt. effective to inhibit interaction of the Monika; Stern, David; Schmidt, Ann Marie ***glycation*** ***end*** product. A-Q-N-I-T-A-R-I-G-E-P-L-V-L-K-C-K-Gpeptide having an amino acid sequence A1 19990503 WO 1998-US21346 19981009 AN 1999:717025 CAPLUS W: AU, CA, JP, MX PRAI US 1997-948131 invention provides ***advanced*** ***glycation*** AU 9897958 invention also amyloid-.beta. Pischetsrieder, LU, MC, NL, amino acid ***cnd*** comprises isolated amt. of PA The Trustees of Columbia University In the City of New York, FILE MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS ENTERED AT 15:59:45 ON 27 ***glycation*** ***endproduct***) for therapeutic use IN Stem, David; ***Yan, Shi Du***; Schmidt, Ann Marie; 158 S RECEPTOR FOR ADVANCED GLYCATION APPLICATION NO (FILE HOME' ENTERED AT 15:59:32 ON 27 DEC 1999) 20 DUP REM L13 (8 DUPLICATES REMOVED) 21 DUP REM L5 (23 DUPLICATES REMOVED) 20 DUP REM L13 (8 DUPLICATES REMOVED) L14 ANSWER 1 OF 20 CAPLUS COPYRIGHT 1999 ACS 7 DUP REM L10 (2 DUPLICATES REMOVED) WO 1998-US21346 24 DUP REM L8 (7 DUPLICATES REMOVED) YOU HAVE REQUESTED DATA FROM 20 ANSWERS ģ TI Ligand-binding site of RAGE (***receptor*** 9 S L8 AND PRESEN7/AB,BI 881 S PRESENILIN-2/AB,BI A1 19990422 72912 S AMYLOID?/AB,BI KIND DATE E STERN DAVID/AU E YAN SHI DU/AU 1999:265908 CAPLUS 28 S L 12 AND L 1 SO PCT Int. Appl., 101 pp. 4 S L 1 AND L 4 31 S L7 AND L1 0 S L1 AND L2 0 L13 AND L2 CONTINUE? Y/(N):y 103 S E2-E3 CODEN: PLXXD2 221 S E3 => d 114 1- bib ab PI WO 9918987 ***advanced*** PATENT NO. 130:301683 => s 113 and 12 **DEC 1999** END%AB,BI English Lamster, Ira DT Patent FAN.CNT => d his USA L15 L14 **L**10 2222 2 222 bovine RAGE, immunostaining of bovine tissues showed RAGE in identified in cultured bovine endothelium, vascular smooth muscle, monocyte-derived macrophages. RAGE antigen was also visualized and in neural tissue where motor neurons, peripheral nerves, and a that RAGE is ***present*** in multiple tissues and suggest the population of cortical neurons were positive. In situ hybridization confirmed the ***presence*** of RAGE mRNA in the tissues, of RAGE-expressing cells in the expanded intima. These results the vasculature as well as neural and cardiac function, prominent potential relevance of AGE-RAGE interactions for modulating Pathological studies of human atherosclerotic plaques showed with rat PC12 pheochromocytes indicated that they provide a in the tissues. Consistent with these data, RAGE antigen and cardiac myocytes as well as in cultures of neonatal rat cardiac vasculature, endothelium, and smooth muscle cells and in neuronal-related cell culture model for examining RAGE involvement in diabetes and in the normal aging process 103 ("YAN SHI"/AU OR "YAN SHI DU"/AU) PROCESSING COMPLETED FOR L13 YAN SHENTSHAN/AU YAN SHI EN/AU YAN SHI FANG/AU YAN SHI MING/AU YAN SHI PIN/AU YAN SHI PING/AU 84 --> YAN SHI DU/AU YAN SHI KAI/AU YAN SHI KUN/AU YAN SHI LEI/AU YAN SHI G/AU YAN SHI/AU 28 L12 AND L1 => e yan shi du/au mononuclear cells => dup rem 113 => s 112 and 11 properties of 8 and studies => s e2-e3

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NY, 10032, USA	end***
SO J. Biol. Chem. (1999), 274(44), 31740-31749 CODEN: IBCHA 2: 18831, 0001, 0058	products (RAGE) is a member of the
PB American Society for Biochemistry and Molecular Biology	surface molecules and engages diver
DT Journal LA English	pathological processes. One class of glycoxidation
	products, termed advanced glycation
glycation end products (AGEs), with the signal-transducing	at sites of oxidant stress in tissues, a
receptor for AGE (RAGE), by administration of the sol.,	amyloldoses. NAGE also functions a
extracellular ligand-binding domain of RAGE, reversed vascular	amyloid beta peptide, known to accuboth
hyperpermeability and summerselenses in disbetic redents. Since	affected brain parenchyma and cereb
the	with these ligands enhances receptor
precise mol. target of sol. RAGE in those settings was not elucidated, we	positive feedback loop whereby recentor occ
tested the hypothesis that predominant specific AGEs within the	RAGE
tissues in disorders such as diabetes and renal failure. Neosilon.	expression, thereby perpetuating and activation.
(carboxymethyl)lysine (CML,) adducts, are ligands of RAGE. We	Sustained expression of RAGE by co
here that physiol. relevant CML modifications of proteins engage	neurons, in
cellular	proximity to these ligands, sets the s
RAGE, thereby activating key cell signaling pathways such as NF-kanna.B	activation and tissue damage. In a model of ac-
and modulating gene expression. Thus, CML-RAGE interaction	associated
triggers	with diabetes in genetically manipul
processes minimately mixed to accelerated vascular and inflammatory	surface RAGE by infusion of a soluble, trun
complications that typify disorders in which inflammation is an established component.	completely summersed formation of vi
The annual contract of the con	atherosclerosis in these diabetic/athe
L14 ANSWER 3 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS DI IPI ICATE 1	RAGE
AN 1999:217923 BIOSIS	occurred in the absence of changes in emphasizing the contribution of a lir
I PREV199900217923	mechanism(s) to atherogenesis, which
TI Activation of ***receptor*** for ***advanced***	interaction of PACE with its licends Entities study
end products: A mechanism for chronic vascular	expression
dysfunction in	has been genetically manipulated an
AU Schmidt, Ann Marie (1); ***Yan, Shi Du**; Wautier,	weight RAGE inhibitors will be required to
Jean-Luc; Stem, David	role for RAGE activation in diabetic vasc
CS (1) Department of Surgery, P and S 17-501, College of Physicians	receptor
and Surgeons of Cohimbia University 630 W 168th St. New York	expression in a microenvironment w
NY, 10032 USA	prolonged receptor stimulation, sugg
SO Circulation Research, (March 19, 1999) Vol. 84, No. 5, pp. 480 407	cellular DACE with its linear to confuse a Con
ISSN: 0009-7330.	KACE with its ligands could be a lat important chronic disorders.
LA English SL English	L14 ANSWER 4 OF 20 BIOSIS COP
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TI Human blood-brain barrier receptors for Alzheimer's amyloid-beta
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               dependent, polarized to the apical side, and saturable with high- and
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  low-affinity dissociation constants of 7.8 +- 1.2 and 52.8 +- 6.2 nM,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          or macrophage scavenger receptor (SR), type A, displayed binding
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 binding to the brain microvascular endothelial cell monolayer was
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    with a Michaelis constant of 45 +- 9 nM, and partially sensitive to
                                                                                                                                                                                                                                                                                                                                                    Schmidt, Ann Marie; Fragione, Blas; Zlokovic, Berislav V. (1) CS (1) USC Sch. Med., 2025 Zonal Ave., RMR 506, Los Angeles, CA 90033 USA
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      LA English
AB A soluble monomeric form of Alzheimer's amyloid-beta (1-40)
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           SO Journal of Clinical Investigation, (Aug. 15, 1998) Vol. 102, No.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        blockade (36%) but not to SR blockade. We conclude that RAGE
                                                                                                                                            Asymmetrical binding, endocytosis, and transcytosis at the apical
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              (sAbeta 1-40) is present in the circulation and could contribute to
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   neurotoxicity if it crosses the brain capillary endothelium, which
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          mediate binding of sAbeta1 40 at the apical side of human BBB,
                                                                                                                                                                                                                                                                                                               Ghiso, Jorge; Kim, Kwang Sik; ***Yan, Shi Du***; Stem,
                                                                                                                                                                                                                                                   AU Mackic, Jasmina B.; Stins, Monique; McComb, J. Gordon;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               ***receptor*** for ***advanced*** ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          intact > 94%. Transcytosis of 1251-sAbetal 40 was time and
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     internalization of 1251-sAbetal 40. The internalized peptide
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     comprises the blood-brain barrier (BBB) in vivo. This study
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  dependent, asymmetrical from the apical to basolateral side,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      human sAbetal 40 using an in vitro model of human BBB.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       respectively. Binding of 1251-sAbetal-40 was inhibited by
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 endothelial binding and transcytosis of a synthetic peptide
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       products) antibody (63%) and by acetylated low density
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     Consistent with these data, transfected cultured cells
                                                                                                                                                                                                                 brain microvascular endothelial cell monolayer.
   AN 1998:437309 BIOSIS
DN PREV199800437309
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     overexpressing RAGE
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     ISSN: 0021-9738.
DT Article
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   lipoproteins (33%)
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              1251-sAbetal-40
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             homologous to
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   4, pp.
734-743.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           characterizes
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         anti-RAGE (
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      temperature
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  ascular lesions. Amelioration of
                                                                                                                                                                                                                                                                                                                                                                                                                         mulate in Alzheimer disease in
                                                                                                                                                                                                                                                                                                                                                    is a signal transduction receptor
                                                                    e immunoglobulin superfamily
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       rosclerotic animals by soluble
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 d with selective low molecular
                                                                                                                                                                                                                                                end products, which occur in
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              ral vasculature. Interaction of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           id- and glycemia-independent
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             ulopathy. However, sustained
                                                                                                                                         se ligands relevant to distinct
RAGE ligands includes
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               es using mice in which RAGE
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  ith a plethora of ligand makes
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            tor contributing to a range of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          ritical target cells, including
nononuclear phagocytes, and
ced*** ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         definitively assign a critical
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  1 plasma lipids or glycemia,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  expression and initiates a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        sted mice, blockade of cell
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             cated form of the receptor
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       apancy triggers increased
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    esting that interaction of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            PYRIGHT 1999 BIOSIS
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      elerated atherosclerosis
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  age for chronic cellular
                                                                                                                                                                                                                                                                                                                     nd in renal failure and
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          th we postulate to be
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         ther wave of cellular
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RAGE is also involved in sAbetal 40 transcytosis.

fluid might provide a means for monitoring neuronal perturbation at These data delineate an inflammatory pathway triggered by contributes to the pathogenesis of AD, and that M-CSF in A-beta on neuronal RAGE. We suggest that M-CSF, thus early stage in AD. diabetic vascular York, NY, USA engagement of compared States of generated Practice), patients neurons survival in AD .5 æ S This interaction may be a useful target for treatment of Alzheimer's neural cells and induces neurotoxic damage typical of Alzheimer's disease. Binding assays for the identification and characterization beta anyloid-binding proteins used to identify the interaction of beta anyloid with RAGE are described. Peptides capable of AU 1997-18327 19970121 DN PREV199799569040
TI Anyloid-beta peptide- ***receptor*** for ***advanced***

Bycation ***endproduct*** interaction elicits Alex, Tourtellotte, Wallace W., Rajavashisth, Tripathi, Chen, Xi, RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, CS (1) Dep. Pathol., Columbia Univ., Coll. Physicians Surgeons, New York, NY ***Yan, Shi Du (1)***; Zhu, Huaijie; Fu, Jin; Yang, Shi APPLICATION NO. AN 1997:525836 CAPLUS DN 127:204001 TI Binding of beta-amyloid protein by an advanced glycation L14 ANSWER 6 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS AB The beta amyloid protein binds to a cell-surface RAGE (L14 ANSWER 5 OF 20 CAPLUS COPYRIGHT 1999 ACS receptor and possible treatment of Alzheimer's disease IN Stern, David; Schmidt, Ann Marie; ***Yan, Shi Du*** PA Trustees of Columbia University, USA for ***advanced*** ***end*** WO 1997-US857 expression of macrophage-colony stimulating factor: A Gabriel C.; Stem, David; Schmidt, Ann Marie Al 19970731 KIND DATE A1 19970820 PRAI US 1996-592070 19960126 pathway in Alzheimer disease. WO 1997-US857 19970121 the interaction are reported. AN 1997:262437 BIOSIS SO PCT Int. Appl., 91 pp. W: AU, CA, JP, MX CODEN: PIXXD2 PATENT NO. PI WO 9726913 MC, NL, PT, SE receptor AU 9718327 proinflammatory DUPLICATE 3 LA English products) in end-product DT Patent FANCNT Godman neuronal DATE disease Ş 6

TI RAGE: a novel cellular ***receptor*** for ***advanced***

L14 ANSWER 9 OF 20 CAPLUS COPYRIGHT 1999 ACS

AN 1996:392878 CAPLUS DN 125:111085

AU Schmidt, Ann Marie; Hori, Osamu; Cao, Rong; ***Yan, Shi

Du***; Brett,

Matsumoto,

glycation ***end*** products

Jerold; Wautier, Jean-Luc; Ogawa, Satoshi; Kuwabara, Keisuke;

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triggering chemotaxis, cell proliferation, increased expression of the
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    macrophage scavenger receptor and apolipoprotein E, and enhanced
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    pathway. AD brain shows increased neuronal expression of M-CSF
                                                                                                                                                                                                                                                     In Alzheimer disease (AD), neurons are thought to be subjected
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                of microglia exposed to A-beta, consistent with pathologic findings
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               ***Endproduct***
SO Proceedings of the National Academy of Sciences of the United
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          with age-matched controls. M-CSF released by A-beta-stimulated
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    proximity to A-beta deposits, and in cerebrospinal fluid from AD
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           there was apprxeq 5-fold increased M-CSF antigen (P lt 0.01),
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             interacts with its cognate receptor, c-fms, on microglia, thereby
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          cell surface receptor for A-beta, induces macrophage-colony
                                                                                                                                                                                                                                                                                                                                     deleterious cytotoxic effects of activated microglia. We
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             factor (M-CSF) by an oxidant sensitive, nuclear factor
                                                                                                                                                                                                                                                                                                                                                                                                                        binding of amyloid-beta peptide (A-beta) to neuronal
                                                                           America, (1997) Vol. 94, No. 10, pp. 5296-5301.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                for ***Advanced*** ***Glycation***
                                                                                                                        ISSN: 0027-8424.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           kappa-B-dependent
                                                                                                                                                                                                                                                                                                                                                                                                                                                                 ***Receptor***
                                                                                                                                                                                                                                                                                                                                                                                  demonstrate that
                                                                                                                                                                  Article
                                                                                                                                                                                                               English
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 (RAGE), a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           stimulating
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AU Schmidt, Ann Marie; ***Yan, Shi Du***; Stem, David M. CS Columbia Univ., New York, NY USA SO Circulation, (10/21/97, 1997) Vol. 96, No. 8 SUPPL., pp. 137.

novel target for therapy of diabetic complications.

binding of AGEs: A

Meeting Info.: 70th Scientific Sessions of the American Heart

Orlando, Florida, USA November 9-12, 1997

ISSN: 0009-7322.

Conference

glycation ***endproducts*** (RAGE) mediates

TI The V-domain of ***receptor*** for ***advanced***

L14 ANSWER 8 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS

AN 1998:15376 BIOSIS DN PREV199800015376

receptor for AGEs (advanced glycation end-products), interaction of

AB A review with 56 refs., including sections on identification of

DT Journal; General Review

with endothelial cell RAGE, and blockade of RAGE as a potential

with mononuclear phagocyte RAGE (receptor for AGEs),

interaction of AGEs

target for

intervention in the development of vascular complications in

AB Exposure of proteins to reducing sugars results in non-enzymic CS Dep. Med. Physiol. Surgery, Columbia Univ., Coll. Physicians with the ultimate formation of advanced glycation end products One means through which AGEs modulate cellular functions is Diabetes Federation Satellite Symposium on "Diabetes and SO Diabetes (1996), 45(Suppl. 3, Proceedings of the 15th CODEN: DIAEAZ; ISSN: 0012-1797 Complications", 1994), S77-S80 Masayasu; Stern, David New York, NY, USA Macrovascular and Surgeons, International DT Journal LA English glycation through TI The ***receptor*** for ***advanced*** ***glycation*** Columbia University College of Physicians and Surgeons, New AU Hori, Osamu; ***Yan, Shi Du***; Schmidt, Ann Marie SO Fundam. Clin. Cardiol. (1997), 29(Endothelium in Clinical ***endproducts*** : implications for the development of L14 ANSWER 7 OF 20 CAPLUS COPYRIGHT 1999 ACS AN 1997:337817 CAPLUS DN 127:15986 311-329 CODEN: FCCAEH; ISSN: 1067-5264 PB Dekker

the MPs infiltrating these lesions. These data indicate that RAGE is least in part, by oxidant stress. AGE-beta-2M reduced cytochrome c CS (1) Dep. Pathol., Columbia Univ., Coll. Physicians Surgeons, 630 ***Yan, Shi Du (1)***; Chen, Xi; Fu, Jin; Chen, Ming, Zhu, a specific, dose-dependent manner (K-d apprxeq 53.5 and apprxeq AGE-beta-2M-mediated monocyte chemotaxis was prevented by of a long-term hemodialysis patient revealed positive staining for for AGEs, or RAGE. 1251-AGE-beta-2M bound to immobilized anti-RAGE IgG. Induction of tumor necrosis factor-alpha (TNF) by MPs exposed to AGE-beta-2M resulted from engagement of John; Migheli, Antonio; Nawroth, Peter, Stern, David; Schmidt, Roher, Alex; Slattery, Timothy; Zhao, Lei; Nagashima, Mariko; TI RAGE and amyloid-beta peptide neurotoxicity in Alzheimer's elaboration of TNF by MPs was inhibited by N-acetylcysteine. patients, a process which may ultimately lead to bone and joint arthropathy of dialysis-related amyloidosis, is mediated by the supernatants were prevented by addition of sRAGE, a process AGE-beta-2M-MP-RAGE interaction likely contributes to the L14 ANSWER 11 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS appearances of TNF transcripts and TNF antigen release into central binding site for AGEs formed in vivo and suggest that with these data, immunohistochemical studies of AGE-laden SO Nature (London), (1996) Vol. 382, No. 6593, pp. 685-691. respectively), a process inhibited in the presence of RAGE inflammatory response in amyloid deposits of long-term 168th St., New York, NY 10032 USA 1996:425030 BIOSIS PREV199699156086 ISSN: 0028-0836. RAGE or to MPs in excess sRAGE or amyloid deposits initiation of an destruction. hemodialysis mediated, at LA English DT Article Ann Marie expression RAGE, as Consistent 81.6 nM, Huaijie; disease. Morser, culture and the Ą ΑU receptor in AGE-mediated perturbation of cellular properties can be AGE-beta-2microglobulin with human mononuclear phagocytes via CS (1) Columbia Univ. Coll. Physicinas and Surgeons, 630 W. 168th ***glycation*** products (AGEs) of the Maillard reaction, known as AGE-beta-2M. substances, expression of heme oxygenase type I, and activation of (RAGE) is such a receptor and is a newly identified member of the (MPs), and vascular smooth muscle cells (SMCs) in both vivo and By using reagents to selectively block access to RAGE, the role of is a form of beta-2microglobulin modified with advanced glycation Binding of AGEs to RAGE results in induction of cellular oxidant oxidant-sensitive pathway: Implications for the pathogenesis of AU Miyata, Toshio; Hori, Osamu; Zhang, Jinghua; ***Yan, Shi An important component of amyloid fibrils in dialysis-related superfamily expressed on endothelial cells (ECs), mononuclear binding to specific cell surface acceptor mols. The receptor for as exemplified by the generation of thiobarbituric acid-reactive endothelial RAGE and result in enhanced oxidant stress in the L14 ANSWER 10 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS SO Journal of Clinical Investigation, (1996) Vol. 98, No. 5, pp. transcription factor NF-kB, with consequences for a range of functions. AGEs on the surface of diabetic red cells enhance ***end*** products (RAGE) is a central mediator of the phagocytes (MPs), cells important in the pathogenesis of the demonstrate here that the interaction of AGE-beta-2M with Ferran, Luis; Iida, Yoshiyasu, Schmidt, An Marie (1) The ***receptor*** for ***advanced*** and S 11-518, New York, NY 10032 USA dialysis-related amyloidosis. AN 1996:480326 BIOSIS DN PREV199699195582 TI The ***receptor*** f dissected in detail. ISSN: 0021-9738. **DUPLICATE 4** interaction of LA English DT Article mononuclear vessel wall. 1088-1094 binding to cellular ş ᅙ ä

inflammatory

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II RAGE: A receptor upregulated in Alzheimer's disease on neurons,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               mediates induction of oxidant stress.

— ***Yan, Shi Du***; Chen, X.; Fu, J.; Chen, M.; Godman, G.;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   ***Yan, Shi-Du (1)***; Chen, Xi; Fu, Jin; Chen, Ming; Zhu,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       Meeting Info.: 48th Annual Meeting of the American Academy of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        SO Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            TI Rage in Alzheimer's disease: A receptor mediating amyloid-beta
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             Zhao, Lei; Nagashima, Mariko; Morser, John; Roher, Alex; Stem,
indirectly by activating microglia. A specific cell-surface acceptor
                                                                   that could focus its effects on target cells has been postulated but
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              and cerebrovascular endothelium that binds amyloid-beta peptide
                                                                                                                                                                                                       ***advanced*** ***glycation*** ***end*** products'
                                                                                                                                          identified. Here we present evidence that the ' ***receptor***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             L14 ANSWER 12 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        L14 ANSWER 13 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS
                                                                                                                                                                                                                                                                                                                                                            and microglia. Increased expression of RAGE in Alzheimer's
                                                                                                                                                                                                                                                                                 such a receptor, and that it mediates effects of the peptide on
                                                                                                                                                                                                                                                                                                                                                                                                                            indicates that it is relevant to the pathogenesis of neuronal
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   SO Neurology, (1996) Vol. 46, No. 2 SUPPL., pp. A276
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          San Francisco, California, USA March 23-30, 1996
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        CS (1) Columbia Univ., New York, NY 10032 USA
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      Washington, D.C., USA November 16-21, 1996
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      peptide-induced activation of microglia.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          AN 1996.347965 BIOSIS
DN PREV199699070321
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  1996:493978 BIOSIS
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             CS New York, NY USA
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 ISSN: 0028-3878
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LA English
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                                                                                                                                                                                                                                                                                                                                                                                                  disease brain
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                                                                                                                                                                                                                                                                                                                               neurons
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because it is neurotoxic-directly by inducing oxidant stress, and

Verstuyft, Judy; Rubin, Edward M.; Liu, Jiankang, Yeo, Helen C.; advanced of the á receptor in AGE-mediated perturbation of cellular properties can be Exposure of proteins to reducing sugars results in nonenzymatic means through which AGEs modulate cellular functions is through (MPs), and vascular smooth muscle cells (SMCs) in both vivo and substances, expression of heme oxygenase type I, and activation of By using reagents to selectively block access to RAGE, the role of transcription factor NF-kappa-B, with consequences for a range of Binding of AGEs to RAGE results in induction of cellular oxidant Matsumoto, Masayasu; Stern, David CS (1) Dep. Physiol., P and S 11-518, Columbia Univ., Coll. Phys. AU Schmidt, Ann Marie (1); Hori, Osamu; Cao, Rong; ***Yan, with the ultimate formation of advanced glycation end products specific cell surface acceptor molecules. The receptor for AGEs superfamily expressed on endothelial cells (ECs), mononuclear as exemplified by the generation of thiobarbituric acid-reactive L14 ANSWER 14 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS AN 1996:375628 BIOSIS DN PREV199699097984 endothelial RAGE and result in enhanced oxidant stress in the L14 ANSWER 15 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS Brett, Jerold; Wautier, Jean-Luc; Ogawa, Satoshi; Kuwabara, functions. AGEs on the surface of diabetic red cells enhance TI A novel cellular ***receptor*** for ***advanced*** W. 168th, New York, NY 10032 USA SO Diabetes, (1996) Vol. 45, No. SUPPL. 3, pp. 577-580. such a receptor and is a newly identified member of the ***glycation*** ***end*** products. ISSN: 0012-1797 immunoglobulin (AGEs). One English DT Article Shi Du*** (RAGE) is vessel wall. binding to Keisuke; glycation cellular ş

An accelerated atherosclerosis model in diabetic apolipoprotein E

AN 1997:2254 BIOSIS DN PREV199799301457 mice: Vascular accumulation of advanced glycation endproducts

(AGEs) and

enhanced expression of their cellular receptor, rage.

AU Park, Lisa (1); Hori, Osamu; ***Yan, Shi Du***; Zou, Yu

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this intervention to interfere with a critical step in the development
                                                                                                                                                                                                                                                                                             inhibition of RAGE may interfere with monocyte chemotaxis and
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the attraction of mononuclear phagocytes into the vessel wall. In
                                                                   cases, the pro-inflammatory effects of AGEs may be inhibited in
                                                                                                                                              presence of RAGE blockade, using either anti-RAGE F(ab')-2 or
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  products (AGEs) has a central role in vessel wall interactions and
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               Schmidt, Ann Marie (1); Hasu, Mirela; Popov, Doina; Zhang,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       ***Receptor*** for ***advanced*** ***glycation***
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     *** Du***; Nagashima, Mariko; Fuentes, Nelson L.; Fuller,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        L14 ANSWER 17 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS
                                                                                                                                                                                                                                                                                                                                                                        into the vessel wall where AGEs deposit/form, suggesting the
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  L14 ANSWER 18 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS
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Meeting Info: 68th Scientific Session of the American Heart
                                                                                                                                                                                                                      RAGE, the extracellular domain of the molecule. These data
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    II Receptor-dependent hyperfibrinogenemia in diabetic mice:
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 Jing Hua; Chen,
Jingxian; ***Yan, Shi Du***; Brett, Jerold; Cao, Rong;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               blockade of the ***receptor*** for ***advanced***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                vascular disease, especially in patients with diabetes.
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DN PREV199698585796
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                                                                                                                                                                                                                                                                                                                                                                                                             potential of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          English
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      ***end*** -products has a central role in mediating the effects of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    ***glycation***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         CS (1) Dep. Physiol., Columbia Univ. Coll. Phys. Surg., New York, NY 10032
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      important in the setting of diabetes mellitus due to hyperglycaemia
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               oxidant stress is the enhanced expression of vascular cell adhesion molecule-1 on the endothelial surface, a critical consequence of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           characteristic of this disorder. Our work has demonstrated that one
                                                             Bruce N.; Andaz, Shahriyour; Stern, David; Schmidt, Ann Marie
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  principal means by which AGEs interact with the vascular wall is
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             is present on the surface of endothelial cells, smooth muscle cells,
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          with RAGE, resulting in the induction of monocyte chemotaxis as
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                                                                                                  CS (1) Columbia Coll. Physicians Surgeons, New York, NY USA
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            advanced glycation end-products on the development of vascular
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     AB Proteins or lipids exposed to aldose sugars undergo initial and
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                irreversible modification resulting in the formation of so-called
                                                                                                                                    SO Circulation, (1996) Vol. 94, No. 8 SUPPL., pp. 136.
Meeting Info: 69th Scientific Sessions of the American Heart
                                                                                                                                                                                                                                                                                                                                                                                                                                               L14 ANSWER 16 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        AU Hori, Osamu (1); ***Yan, Shi Du***; Ogawa, Satoshi;
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                interaction with their cellular receptor, the ***receptor***
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         SO Nephrology Dialysis Transplantation, (1996) Vol. 11, No.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       glycation end-products (AGEs). AGEs are postulated to be
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       Matsumoto, Masayasu; Stem, David; Schmidt, Ann Marie
                                                                                                                                                                                                                                                       New Orleans, Louisiana, USA November 10-13, 1996
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   AN 1997:128146 BIOSIS
DN PREV199799419959
TI The ***receptor*** for ***advanced***
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                                                                                                                                                                                                                                                                                                                                  Conference; Abstract
                                                                                                                                                                                                                                                                                         ISSN: 0009-7322
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          diabetes mellitus.
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 DUPLICATE 6
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           SUPPL. 5, pp.
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ISSN: 0027-8424.	Association	atherocclerotic plannes showed infiltration of DAA To A
JT Article	Dallas, Texas, USA November 14-17, 1994	cells in
A English	ISSN: 0009-7322.	the expanded intima. These results indicate that RAGE is present
AB I he extended interaction of aldoses with proteins or lipids results	DT Conference	s
nonenzymatic glycation and oxidation, ultimately forming AGEs,	LA English	multiple tissues and suggest the potential relevance of AGE-RAGE
in the second se	L14 ANSWER 20 OF 20 BIOSIS COPYRIGHT 1999 BIOSIS	neural and cardiac function, prominent areas of involvement in
diabetic vascular complications. We show that ACC alternia	DUPLICATE 8	diabetes
the	AIN 1994:334438 BIOSIS DN PREV199497367438	and in the normal aging process.
intravascular space interacts with the vessel wall via binding to an	TI Survey of the distribution of a newly characterized	
integral membrane protein, receptor for AGE (RAGE), a member	***receptor*** for	=> e wolozin benjamin/au
if the immunoal challe and comits and the contract of the cont	***advanced*** ***glycation*** ***end*** products in	
unnimoglobum supenamny, resuning in clearance from the Jasma and	tissues.	en -
induction of interleukin 6 mRNA. Intravenously infused 1251-AGE	Au Dieu, Jeroid, Schimidi, Ann Marie, *** I an, Shi Du***; Zou, Yii Shan:	- 2
lbumin	Weideman, Eljott: Pinsky, David: Nowygrod Roman: Neener	
showed a rapid phase of plasma clearance with deposition in	Michael;	, 1 w
everal	Przysiecki, Craig, Shaw, Alan, Migheli, Antonio, Stern, David (1)	
organs, respire removat of 1231-AGE atomin from the prasma was revented by	C3 (1) Dep. Physiology, P and S 11-518, Columbia Univ., Coll P and S 530	- .
administration of a soluble, truncated form of RAGE, which	West 168th Street New York NV 10032 119A	E8 1 WOLOGSZUK RAU
locked binding	SO American Journal of Pathology, (1993) Vol. 143, No. 6, pp.	
of 1251-labeled AGE albumin to cultured endothelial cells and	1699-1712.	. 7
nononuclear	ISSN: 0002-9440.	
phagocytes, as well as by pretreatment with anti-RAGE IgG.		
studies with AGE albumin-colloidal oold conjugate perfised in	LA English AB Advanced chronism and modelism (ACEA) the Gard and details	=>sel-c4
itu showed	nonenzymatic	1 16 61 ("WOLOZIN BENI"/ALLOB "WOLOZIN BENIA)
that in murine coronary vasculature this probe was taken up by	glycation and oxidation of proteins, are found in the plasma and	0
ndothelial	accumulate in the tissues during aging and at an accelerated rate in	N"/AU OR "WOLOZIN BENJAMIN L"/AU)
plasmatemmal vestcles followed by transport either to the himming!	diabetes. A novel integral membrane protein, termed receptor for	
stutinisal stuttace of by accumulation in lysosomes. Consequences of	AUE (PACE) forms a control and of the call suches him dies size for	=> s 6 and
GE-RAGE	(ICACE), forms a central part of the cent surface origing site for AGEs.	117 0115 430011
interaction included induction of interleukin 6 mRNA expression in	Using monospecific, polyclonal antibody raised to human	
nice.	recombinant and	=> s 116 and 12
These data indicate that RAGE mediates the interaction of AGEs	bovine RAGE, immunostaining of bovine tissues showed RAGE in	
usi are Vessel wall hoth for removal of these physical motains from the	the the second s	L18 4L16ANDL2
lasma	vasculature, citabaticituri, ana sinooti muscie cens and m mononuclear cells	118 mar an b <=
and for changes in gene expression.	in the tissues. Consistent with these data, RAGE antigen and	
14 ANSWER 19 OF 20 BIOSIS CORVEIGHT 1000 BIOSIS	mRNA were	CESS
N 1995;8108 BIOSIS	and	LIS 3 DUP REM LIS (1 DUPLICATE REMOVED)
N PREV199598022408	monocyte-derived macrophages. RAGE antigen was also visualized	=> d 1- bib ab
1 The mononuclear phagocyte interaction site of	in bovine	
modified by glycation is the ***recentor*** for	cardiac myocytes as well as in cultures of neonatal rat cardiac	YOU HAVE REQUESTED DATA FROM 3 ANSWERS -
advanced*	and in neural tissue where motor neurons, peripheral nerves, and a	CONTINUES IN(N);
glycation ***endproducts***	population of cortical neurons were positive. In situ hybridization	
VO Schmidt, Ann Marie (1); Hori, Osamu; *** Yan, Shi Du***; Sawa.	confirmed the presence of RAGE mRNA in the tissues, and studies	
Satoshi; Stern, David; Miyata, Toshio	PC12 pheochromocytes indicated that they provide a	AN 1998:224560 BIOSIS
S (1) Columbia Univ., New York, NY USA	neuronal-related cell	TI Regulation of apoptosis by presentlin 1.
O Circulation, (1994) Vol. 90, No. 4 PART 2, pp. 1233. Meeting Info: 67th Scientiffs Services of the American II.	culture model for examining RAGE expression. Pathological	AU ***Wolozin, Benjamin (1)***; Alexander, P., Palacino, J.
Meeting tho otal ocientife oessions of the American near	studies of human	CS (1) Dep. Pharmacol., Loyola Univ. Medical Cent., Build. 102,

Room 3634,	hydrogen	DEC 1999
2160 South First Ave., Maywood, IL 60153 USA SO Neurobiology of Aging (Ign.: Feb. 1908) Vol. 19 No. 1	peroxide or the free radical MPP+. Thus, the PS2 gene is required	L1 158 S RECEPTOR FOR ADVANCED GLYCATION
SUPPL, pp.	some forms of cell death in diverse cell types, and its function is	END'//AB,BI 1.2 881 S PRESENTI IN 2/AB BI
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AB Familial Alzheimer's disease is transmitted as an autosomal	AN 1997.31943 BIOSIS	
dominant		(4
disorder and, in 5-10% of the cases, is caused by mutations in the	TI Participation of ***Presentlin*** ***2*** in apoptosis:	
regions of two homologous genes Presentlin 1 and 2 (PQ) and	Enhanced book activity confound by an Alaksian activity	(1
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Previously, we have shown that PS2, a homolog of PS1, regulates	2	EII / DOF NEM LIU (2 DOPLICATES NEMOVED) F YAN SHI DII/AII
apoptosis	Ganjei, J. Kelly, Lacana, Emanuela, Sunderland, Trey, Zhao, Boyu;	L12 103 S B2-E3
induced in neurons by trophic withdrawal or Abeta, and in T-cells	Kusiak,	
by Fass	John W.; Wasco, Wilma; D'Adamio, Luciano	
ingain. We now repoil that Pol also regulates apoptosis, both wild-tyne	CS (1) Dep. Pharmacol., Loyola Univ. Med. Cent., 2160 South First	L15 0 S L13 AND L2
and the H115Y mutant form of PS1 enhance Fas-mediated	Managed II 60152118A	
apoptosis in Jurkat	SO Science (Washington D.C.) (1996) Vol. 274 No. 5203 m.	L16 61 S E1-E4
cells. We also observed that wild-type and the H115Y mutant form	1710-1713.	L17 US L16 AND L1
ofPS1	ISSN: 0036-8075.	
differentially regulate Jun Kinase, an important enzyme regulating		
apoptosis.	LA English	û
L19 ANSWER 2 OF 3 CAPLUS COPYRIGHT 1999 ACS	AB Overexpression of the familial Alzheimer's disease gene	a manufacture of the second of
AN 1996.761948 CAPLUS	***2*** (PS2) in nerve growth factor-differentiated PC12 cells	rossing on or o in
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Katsunori, Lacana, Emanuela, D'Adamio, Luciano	arryloid precursor protein-expressing PC12 cells. The anontotic	\ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \
CS NIAID, Natl Inst. Health, Bethesda, MD, 20892, USA	cell death	
SO J. Biol. Chem. (1996), 271(49), 31025-31028	induced by PS2 protein was sensitive to pertussis toxin, suggesting	COST IN U.S. DOLLARS SINCE FILE TOTAL
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	associated what regulated registrates a disease was found to generate 8	DISCOINT AMOINTS GOR OTIAT TEVING ACCOUNTS
AB ALC-3, a truncated mouse homolog of the chromosome 1	molecule with enhanced basal apoptotic activity. This gain of	SINCE FILE TOTAL
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